Lydia Aguilar-Bryan, M.D., Ph.D.

Associate Professor of Medicine and Molecular and Cell Biology
Division of Diabetes, Endocrinology and Metabolism
Baylor College of Medicine

Research Interests

We have for several years been interested in understanding the molecular mechanisms by which glucose stimulates insulin secretion, in particular the role that ATP-sensitive K^+ channels (K_{ATP}) play in this process. Our laboratory cloned the high-affinity sulfonylurea receptor (SUR1), the regulatory subunit of this channel, and we have been screening for mutations in patients with insulin secretory abnormalities, such as neonatal diabetes, IDDM, NIDDM, and Hyperinsulinemic Hypoglycemia (HI), and doing structure-function studies. It has become clear that mutations in SUR1, which are responsible for ~50% of the cases of HI, result in more than an ion channel defect; to this end, we have been identifying other players that potentiate or inhibit glucose-stimulated insulin secretion and how they are altered as a result of the lack of this potassium channel activity.

It has also become clear that the Sur1 KO mice that we generated, with the idea of understanding better the HI phenotype, present a very interesting resistant phenotype towards different forms of stress (STZ, hypoglycemia, kainic acid, and cytokines). Towards this end we are working on the role that these channels may play in the apoptotic and/or preconditioning process effect in pancreatic beta cells and in the brain.

We are also using this model to better understand the role that these channels may play in the counter-regulatory response to hypoglycemia, the effect of hormones whose action is determined by G-protein coupled receptors and the effect of GLP-1 in Islet Biology.

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